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CORRELATION OF EPICARDIAL ADIPOSE TISSUE THICKNESS WITH DAS28 SCORE IN PATIENTS WITH RHEUMATOID ARTHRITIS AT A TERTIARY CARE HOSPITAL IN CHENNAI, INDIA

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ABSTRACT

Background: Rheumatoid arthritis (RA) is a chronic systemic inflammatory disease strongly associated with premature cardiovascular disease (CVD) and excess cardiovascular mortality. Epicardial adipose tissue (EAT), a metabolically active visceral fat depot in direct contact with the myocardium and coronary vessels, has emerged as a promising imaging biomarker of subclinical cardiovascular risk. However, its relationship with disease activity in RA remains under-explored in South Asian populations.

Objective: To compare echocardiographic EAT thickness between patients with RA and healthy controls, and to evaluate its correlation with disease activity as assessed by the Disease Activity Score in 28 joints (DAS28).

Methods: This hospital-based, case–control study was conducted at Sree Balaji Medical College and Hospital, Chennai, between June 2023 and June 2024. Forty patients (aged 18–40 years) with stable RA diagnosed according to the revised American College of Rheumatology (ACR) criteria and forty age- and sex-matched healthy controls were recruited. Individuals with traditional cardiovascular risk factors, coronary artery disease, or other systemic conditions influencing EAT were excluded. EAT thickness was measured on the free wall of the right ventricle using 2D transthoracic echocardiography from the parasternal long-axis view at end-diastole. Disease activity was quantified using the DAS28-ESR. Comparisons were made using independent-samples t-tests and one-way ANOVA; correlations were assessed with Pearson's coefficient. A p-value <0.05 was considered statistically significant.

Results: The mean EAT thickness was significantly greater in patients with RA than in controls (5.76 ± 1.17 mm vs. 3.84 ± 0.69 mm; $p<0.001$). Among patients with RA, 10% were in remission, 35% had low, 32.5% had moderate, and 22.5% had high disease activity. EAT thickness increased progressively across disease activity categories, from 4.23 ± 0.31 mm in remission to 7.51 ± 0.44 mm in the high-activity subgroup ($p<0.001$). No significant difference in EAT was observed by disease duration ($p=0.612$) or treatment status ($p=0.25$).

Conclusion: Patients with RA have significantly greater EAT thickness than healthy controls, and EAT thickness correlates directly with DAS28-defined disease activity. Two-dimensional echocardiographic measurement of EAT is a simple, low-cost, and non-invasive tool that may be integrated into routine cardiovascular risk stratification of patients with RA and may guide the earlier and more intensive use of disease-modifying therapy.

KEYWORDS: Rheumatoid arthritis; Epicardial adipose tissue; DAS28; Cardiovascular risk; Echocardiography; Systemic inflammation.

1. INTRODUCTION

Rheumatoid arthritis (RA) is a chronic systemic autoimmune disorder characterized by symmetric, erosive polyarthritis that predominantly affects the small joints of the hands and feet. Its global prevalence is estimated at 0.5–1% of the adult population, with a marked female predominance of approximately 2.5:1 and a peak incidence between the fourth and fifth decades of life.[1,2]

Beyond its articular manifestations, RA is now firmly recognized as an independent cardiovascular risk state. Cardiovascular disease (CVD) accounts for nearly half of all deaths in patients with RA, and the excess risk is only partially explained by traditional cardiovascular risk factors such as hypertension, dyslipidaemia, obesity, and diabetes mellitus.[3–5] Persistent systemic inflammation, mediated by cytokines such as tumour necrosis factor-α (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP), accelerates endothelial dysfunction and atherogenesis, often producing subclinical vascular disease long before overt cardiovascular events become apparent. Early detection of this subclinical burden is therefore a priority in contemporary RA management.

Epicardial adipose tissue (EAT) is a unique visceral fat depot located between the myocardium and the visceral pericardium, sharing an unobstructed microcirculation with the underlying myocardium and coronary arteries.

Under physiological conditions EAT exerts local metabolic and thermogenic functions, but in the setting of chronic inflammation it transitions to a pro-atherogenic phenotype, secreting adipokines and pro-inflammatory mediators that promote coronary atherosclerosis, myocardial fibrosis, and diastolic dysfunction.[6] Two-dimensional (2D) transthoracic echocardiography allows reliable, reproducible, and inexpensive measurement of EAT thickness on the free wall of the right ventricle, offering a practical alternative to cardiac magnetic resonance imaging (MRI) or computed tomography (CT).

Disease activity in RA is most commonly quantified using the Disease Activity Score in 28 joints (DAS28), a composite index with a reported sensitivity and specificity of approximately 90% and 82% respectively when compared with other activity scores.[7] Because the same inflammatory milieu that drives synovitis is also implicated in EAT expansion, a direct relationship between DAS28 and EAT thickness is biologically plausible. However, data from Indian and South Asian cohorts remain limited, and the extent to which EAT tracks with disease activity in young, otherwise low-risk patients has not been well characterized.

The present study was therefore designed to (i) compare echocardiographic EAT thickness between patients with RA and age- and sex-matched healthy controls at a tertiary care hospital in Chennai, India, and (ii) evaluate the correlation between EAT thickness and DAS28-defined disease activity. We hypothesized that EAT thickness would be significantly greater in patients with RA than in controls, and that it would increase progressively with worsening disease activity, thereby supporting its use as an accessible surrogate marker of subclinical cardiovascular risk.

2. MATERIALS AND METHODS

2.1. Study design and setting

This was a single-centre, cross-sectional, case–control study conducted in the Department of General Medicine in collaboration with the Department of Rheumatology at Sree Balaji Medical College and Hospital, Chennai, India, between June 2023 and June 2024. The study protocol was reviewed and approved by the Institutional Ethics Committee, and the study was carried out in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from every participant prior to enrolment.

2.2. Study participants

A total of eighty participants were recruited by consecutive sampling: forty patients with stable RA attending the Rheumatology outpatient department and forty apparently healthy volunteers attending the General Medicine outpatient department for routine health check-ups, matched for age and sex. RA was diagnosed according to the revised American College of Rheumatology (ACR) classification criteria.

2.3. Inclusion criteria

Patients aged 18–40 years with stable RA fulfilling the revised ACR criteria, who had no acute intercurrent illness at the time of assessment, were eligible for inclusion. Healthy controls in the same age group without any known chronic disease were recruited from the general outpatient department.

2.4. Exclusion criteria

To minimize the influence of confounders known to alter EAT thickness, participants were excluded if they had any of the following: established coronary artery disease, valvular heart disease, previous heart failure or liver disease; systemic hypertension; diabetes mellitus or a fasting plasma glucose >126 mg/dL; hypothyroidism or hyperthyroidism; chronic kidney disease or renal failure; a history of cerebrovascular accident; body mass index (BMI) >25 kg/m²; current smoking or regular alcohol consumption; or any other coexisting autoimmune disease.

2.5. Echocardiographic assessment of epicardial adipose tissue

All participants underwent transthoracic 2D and Doppler echocardiography, including tissue Doppler imaging, after a resting period of 15 minutes, using a 3.5 MHz transducer. Recordings were obtained in the left lateral decubitus position from standard parasternal and apical views during quiet respiration, capturing both inspiratory and end-expiratory phases. M-mode, pulse-wave, and continuous-wave Doppler tracings were acquired for each case. All measurements were performed in accordance with the recommendations of the American Society of Echocardiography by a single experienced cardiologist blinded to the clinical group of the participant.

EAT was defined as the echo-free space between the outer wall of the myocardium and the visceral layer of the pericardium. EAT thickness was measured on the free wall of the right ventricle from the parasternal long-axis view, perpendicular to the right ventricular free wall at end-diastole, using the aortic annulus as an anatomical reference. The mean of measurements obtained from three consecutive cardiac cycles was recorded as the final EAT thickness for each participant.

2.6. Laboratory investigations

Venous blood samples were collected in the morning after an 8-hour overnight fast. Fasting plasma glucose and lipid profile were measured by standard enzymatic methods, and thyroid function tests (TSH, free T4, free T3) by immunoassay. In addition, all participants underwent complete blood count with erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), rheumatoid factor (RF), antinuclear antibody (ANA), liver function tests, and renal function tests using standard automated laboratory platforms.

2.7. Assessment of disease activity

Disease activity in RA was quantified using the DAS28-ESR, a composite score derived from the tender joint count (out of 28 joints), the swollen joint count (out of the same 28 joints), the ESR, and the patient's global health assessment on a 0–100 mm visual analogue scale. Patients were then categorized into four groups according to

the standard cut-offs: remission (DAS28 ≤2.6), low (>2.6 to ≤3.2), moderate (>3.2 to ≤5.1), and high (>5.1) disease activity.

2.8. Statistical analysis

Data were entered in a Microsoft Excel spreadsheet and cross-verified, then analyzed using IBM SPSS Statistics version 25. Categorical variables were summarized as frequencies and percentages, and continuous variables as mean ± standard deviation. Between-group comparisons of continuous variables were performed using the independent-samples t-test, and comparisons across the four DAS28 activity categories using one-way analysis of variance (ANOVA). The relationship between EAT thickness and other continuous variables was examined using Pearson's correlation coefficient. A two-sided p-value <0.05 was considered statistically significant.

3. RESULTS

3.1. Baseline demographic characteristics

The demographic profile of the case and control groups was closely matched (Table 1, Figure 1). The majority of participants in both groups belonged to the 36–40 year age band (60% of cases vs. 52.5% of controls). Younger age bands were represented in comparable proportions: 22.5% of cases and 12.5% of controls in the 30–35 year band and 17.5% of cases and 27.5% of controls in the 26–30 year band. Sex distribution was identical in the two groups, with 50% men and 50% women in each.

Table 1. Age and sex distribution of the study population (n = 80)

Variable	Category	Cases (n=40), n (%)	Controls (n=40), n (%)
Age (years)	<25	0 (0.0)	3 (7.5)
	26–30	7 (17.5)	11 (27.5)
	30–35	9 (22.5)	5 (12.5)
	36–40	24 (60.0)	21 (52.5)
Sex	Male	20 (50.0)	20 (50.0)
	Female	20 (50.0)	20 (50.0)

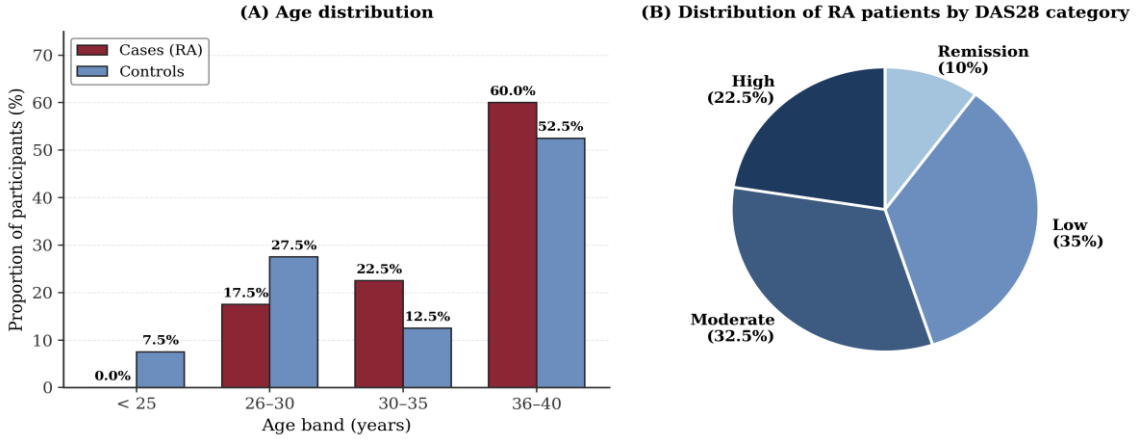

Figure 1. Baseline distribution of the study population. (A) Age-band distribution of cases (RA) and controls; (B) proportional distribution of the 40 patients with RA across the four DAS28-ESR disease activity categories.

3.2. Clinical profile of patients with rheumatoid arthritis

Among the 40 patients with RA, a broad spectrum of disease activity was captured. Based on the DAS28-ESR, 4 patients (10%) were in remission, 14 (35%) had low disease activity, 13 (32.5%) had moderate disease activity, and 9 (22.5%) had high disease activity (Figure 1B). Twenty-eight patients (70%) were already receiving disease-modifying antirheumatic drug (DMARD) therapy, whereas the remaining 12 (30%) were treatment-naïve at recruitment. Chronic disease exposure predominated in the cohort: 31 patients (77.5%) had a disease duration exceeding 5 years, while only 9 (22.5%) had a shorter duration (Table 2).

Table 2. Clinical profile of patients with rheumatoid arthritis (n = 40)

Variable	Category	n (%)
DAS28 disease activity	Remission	4 (10.0)
	Low activity	14 (35.0)
	Moderate activity	13 (32.5)
	High activity	9 (22.5)
Treatment status	On DMARD therapy	28 (70.0)
	Treatment-naïve	12 (30.0)
Disease duration (years)	> 5	31 (77.5)
	≤ 5	9 (22.5)

3.3. Epicardial adipose tissue thickness in RA versus controls

The mean EAT thickness was significantly greater in patients with RA than in healthy controls (5.76 ± 1.17 mm vs. 3.84 ± 0.69 mm; p<0.001), representing an approximately 50% relative increase in the case group (Table 3, Figure 2). This finding confirms a strong association between the presence of RA and increased echocardiographic EAT thickness, independent of established cardiovascular risk factors, which were excluded by design.

Table 3. Comparison of epicardial adipose tissue thickness between cases and controls

Parameter	Cases (n=40)	Controls (n=40)	p-value
EAT thickness (mm), mean ± SD	5.76 ± 1.17	3.84 ± 0.69	**<0.001**

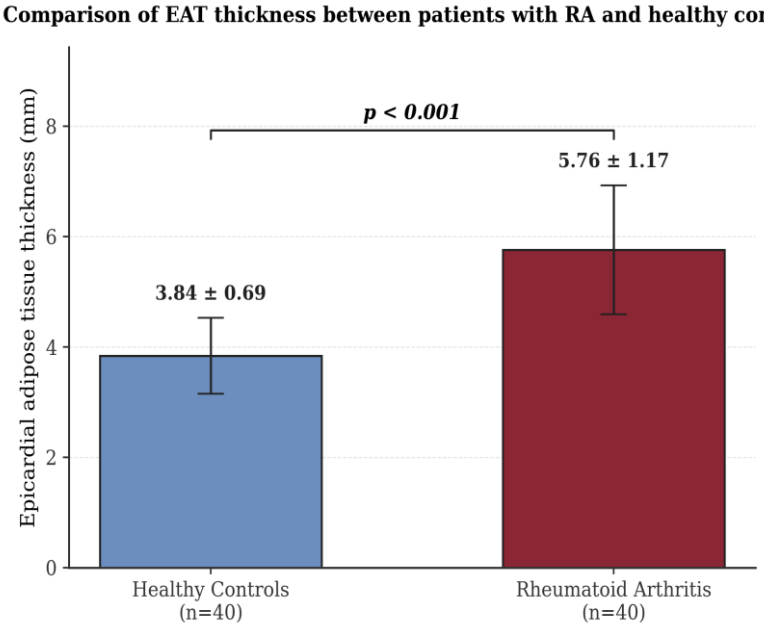

Figure 2. Comparison of mean epicardial adipose tissue (EAT) thickness between patients with rheumatoid arthritis (RA) and healthy controls. Bars represent mean ± standard deviation. EAT thickness was significantly greater in patients with RA (independent-samples t-test, p<0.001).

3.4. EAT thickness by disease activity, duration, and treatment status

EAT thickness increased in a clear stepwise manner across the four DAS28 activity categories (Table 4, Figure 3). Patients in remission had the thinnest EAT (4.23 ± 0.31 mm), followed by those with low activity (4.96 ± 0.48 mm) and moderate activity (5.85 ± 0.49 mm), while patients with high disease activity had the greatest EAT thickness (7.51 ± 0.44 mm). The overall difference across the four groups was statistically highly significant (p<0.001), consistent with a direct dose–response relationship between disease activity and EAT accumulation.

Table 4. Epicardial adipose tissue thickness by disease duration, DAS28 activity, and treatment status

Variable	Category	EAT (mm), mean ± SD	p-value
Disease duration	≤ 5 years	5.81 ± 1.23	0.612
	> 5 years	5.58 ± 0.99	
DAS28 category	Remission	4.23 ± 0.31	**<0.001**
	Low activity	4.96 ± 0.48	
	Moderate activity	5.85 ± 0.49	
	High activity	7.51 ± 0.44	
Treatment status	On DMARD therapy	5.59 ± 1.07	0.25
	Treatment-naïve	6.14 ± 1.34	

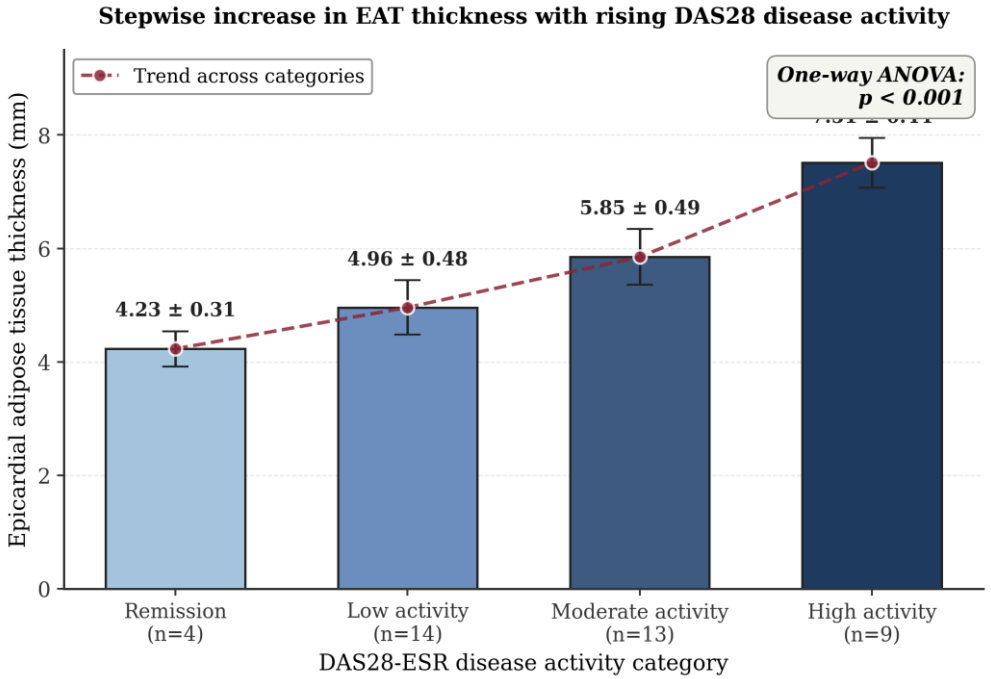

Figure 3. Stepwise increase in epicardial adipose tissue (EAT) thickness with rising DAS28-ESR disease activity in the 40 patients with rheumatoid arthritis. Bars represent mean ± standard deviation; the dashed line highlights the near-linear rise in mean EAT thickness across categories. The between-group difference was highly significant (one-way ANOVA, p<0.001).

In contrast, no significant difference in EAT thickness was observed between patients with a disease duration ≤5 years and those with a duration >5 years (5.81 ± 1.23 mm vs. 5.58 ± 0.99 mm; p=0.612), nor between treatment-naïve patients and those already on DMARD therapy (6.14 ± 1.34 mm vs. 5.59 ± 1.07 mm; p=0.25) (Figure 4). These findings suggest that current disease activity, rather than cumulative disease exposure or ongoing therapy, is the principal correlate of EAT thickness in this cohort.

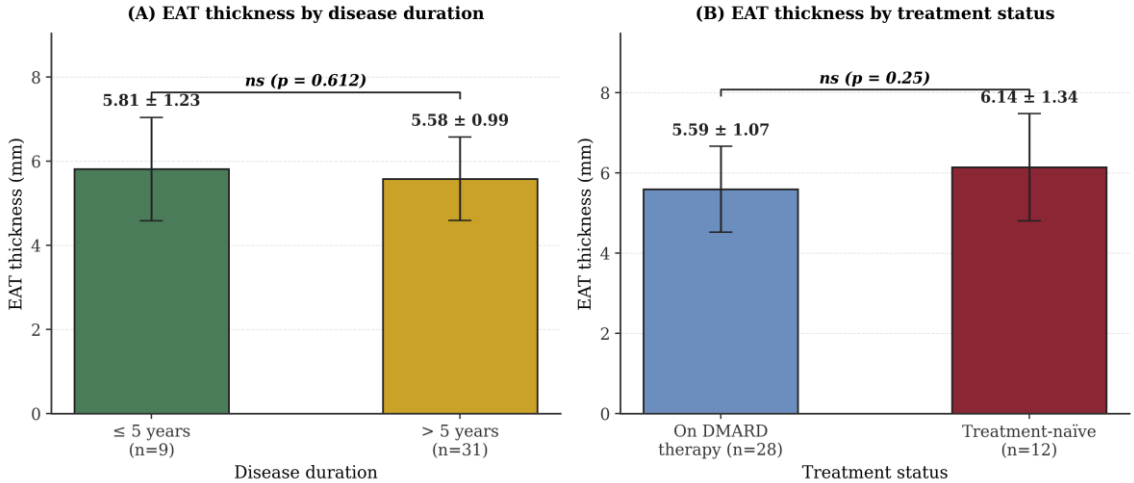

Figure 4. Non-significant subgroup analyses within patients with rheumatoid arthritis. (A) EAT thickness by disease duration (≤ 5 vs. > 5 years, p=0.612). (B) EAT thickness by treatment status (on DMARD therapy vs. treatment-naïve, p=0.25). Bars represent mean ± standard deviation; "ns" = non-significant.

4. DISCUSSION

In this case–control study of young adults with stable RA at a tertiary care centre in Chennai, we observed two principal findings. First, echocardiographic EAT thickness was significantly greater in patients with RA than in age- and sex-matched healthy controls (5.76 ± 1.17 mm vs. 3.84 ± 0.69 mm; p<0.001) (Figure 2), even after excluding participants with traditional cardiovascular risk factors. Second, EAT thickness rose progressively across DAS28 activity categories, from a mean of 4.23 mm in patients in remission to 7.51 mm in those with high disease activity (p<0.001) (Figure 3). Together, these results support the hypothesis that active systemic inflammation in RA is a key driver of epicardial fat accumulation and, by extension, of subclinical cardiovascular risk.

The observation that EAT thickness did not vary significantly by age or sex within our cohort suggests that, in a relatively young population from which conventional cardiometabolic risk factors have been excluded, demographic variables contribute little to inter-individual variation in EAT. Similarly, disease duration (p=0.612) and treatment status (p=0.25) were not significantly associated with EAT thickness (Figure 4). This is an important observation: it implies that the short-term inflammatory burden reflected by DAS28 may be a more sensitive determinant of epicardial fat than either cumulative disease exposure or the mere presence of DMARD therapy. Because 70% of our patients were already on treatment, this may also reflect residual inflammation despite conventional therapy, and highlights the possibility that standard DMARDs do not uniformly translate into reduced epicardial fat deposition.

Our results are broadly concordant with previously published work in RA and other systemic inflammatory diseases. Keleşoğlu Dinçer et al. reported significantly greater EAT thickness in patients with RA than in controls, along with a significant correlation between EAT and DAS28, and identified an EAT cut-off of ≥6.4 mm as a marker of higher disease activity and CRP levels.[6] Wang et al. similarly demonstrated significantly thicker EAT in patients with RA and showed its association with impaired left ventricular systolic function using speckle-tracking echocardiography.[10] Saha et al. found higher EAT, greater left ventricular mass, and more frequent diastolic dysfunction in RA patients compared with controls, with DAS28, disease duration, RF, ESR, and high-sensitivity CRP all associated with higher EAT.[9] Farhat et al., in a systematic review, further confirmed the elevated cardiovascular risk profile of patients with RA and the role of EAT as a clinically relevant surrogate marker.[8]

CVD accounts for approximately 50% of all deaths in patients with RA,[5] and matched-cohort data such as those reported by Argnani et al. within the Italian RECORD registry confirm that this risk exceeds that of the general population even after adjustment for classical factors.[11] The extent to which this excess is attributable to systemic inflammation rather than to traditional risk factors remains an active area of investigation. Our data—in which patients with traditional risk factors were excluded by design—support an important, independent contribution of inflammation itself, mediated in part through expansion of the epicardial fat depot.

The biological plausibility of this association is well established. TNF-α, a central cytokine in RA pathogenesis, promotes adipose tissue inflammation and endothelial dysfunction, and TNF-α inhibitors have been shown to reduce EAT thickness and to improve cardiovascular outcomes in RA. Lima-Martínez et al. demonstrated that RA patients receiving TNF-α inhibitors had significantly lower EAT than those not receiving these agents,[3] while Gastaldelli et al. linked EAT and ectopic fat to hypertension and coronary artery disease.[12] Bal et al. reported a similar association between EAT and RA in a Turkish cohort of 31 patients,[13] and increased EAT has also been documented in ankylosing spondylitis (Resorlu et al.),[14] systemic lupus erythematosus (Lipson et al.),[15] and other systemic inflammatory conditions leading to subclinical atherosclerosis (Fatma et al.).[16] Collectively, these observations support the view that the inflammatory milieu itself, rather than any single disease-specific mechanism, drives EAT expansion.

Two further studies are particularly relevant to our findings. Alpaydın et al. investigated newly diagnosed RA patients free of conventional cardiovascular risk factors and reported that higher DAS28 scores were independently associated with both increased EAT thickness ($r = 0.365$, $p = 0.02$) and impaired left ventricular diastolic function (odds ratio 2.556, $p = 0.009$), providing a mechanistic link between disease activity and subclinical myocardial dysfunction.[17] Ilgun et al. also demonstrated significantly greater EAT in patients with RA than in controls (0.68 ± 0.16 cm vs. 0.40 ± 0.11 cm; $p<0.001$), although in their cohort correlations with inflammatory markers and disease duration were not significant, suggesting that EAT accumulation can occur even in the absence of overt biochemical inflammation.[18]

At a conceptual level, Tarsitano et al., synthesizing 50 studies and more than 10,000 patients, emphasized the utility of EAT as a non-invasive imaging biomarker for cardiometabolic risk stratification in chronic inflammatory diseases.[19] Packer proposed that EAT acts as a pathogenic transducer of inflammation and metabolic stress on the myocardium, transitioning from a protective to a harmful phenotype under chronic inflammatory conditions such as RA and releasing adipokines that promote myocardial fibrosis and diastolic dysfunction.[20] Nagy et al. similarly outlined the mechanistic role of EAT in local inflammation and atherogenesis, endorsing its clinical relevance as an emerging cardiovascular risk indicator.[21]

Set against this background, our study contributes fresh Indian data confirming the diagnostic and prognostic relevance of EAT in RA. The strength of the association between DAS28 and EAT observed in our cohort reinforces the clinical importance of achieving tight disease control. Early initiation of DMARD therapy—including biologic agents where indicated—may not only prevent joint damage but also mitigate cardiovascular mortality by limiting systemic inflammation and, secondarily, EAT expansion. Although cardiac MRI remains the gold standard for quantifying EAT, 2D transthoracic echocardiography provides a practical, low-cost, and radiation-free alternative that is well suited to routine outpatient practice, particularly in resource-constrained settings.

5. LIMITATIONS

Several limitations of this study warrant acknowledgement. First, it was a single-centre study with a relatively modest sample size ($n = 80$), which limits the generalizability of the findings to broader populations. Second, the cross-sectional design precludes any inference regarding causality: whether reducing EAT thickness translates

into measurable cardiovascular benefit in RA remains to be tested in longitudinal and interventional studies. Third, although key confounders were excluded by design, unmeasured subclinical factors such as dietary habits, physical activity, cumulative glucocorticoid exposure, and specific DMARD subclasses may have influenced EAT thickness. Finally, echocardiographic measurement of EAT, while validated and reproducible, is inherently operator-dependent; blinded assessment by a single experienced cardiologist mitigated but did not eliminate this constraint. Larger, multicentre, prospective studies with longitudinal follow-up and comparison against cardiac MRI are needed to confirm and extend our observations.

6. CONCLUSION

Patients with rheumatoid arthritis have significantly greater echocardiographic epicardial adipose tissue thickness than healthy controls, and this difference is closely linked to disease activity as measured by the DAS28-ESR. EAT thickness rose progressively from patients in remission to those with high disease activity, whereas neither disease duration nor treatment status showed a significant independent effect. These findings support the integration of routine echocardiographic EAT measurement into cardiovascular risk stratification in patients with RA, particularly those with active disease, and reinforce the rationale for early, sustained control of systemic inflammation to reduce long-term cardiovascular mortality in this high-risk population.

DECLARATIONS

Ethical approval and consent to participate

The study protocol was approved by the Institutional Ethics Committee of Sree Balaji Medical College and Hospital, Chennai. The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

Conflict of interest

The authors declare that they have no competing interests.

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Authors' contributions

VS conceived the study, collected the clinical data, and drafted the manuscript. APA supervised the study, contributed to the design and interpretation, and critically revised the manuscript. Both authors read and approved the final version.

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REFERENCES

1. Guo Q, Wang Y, Xu D, Nossent J, Pavlos NJ, Xu J. Rheumatoid arthritis: pathological mechanisms and modern pharmacologic therapies. Bone Res. 2018;6:15. https://doi.org/10.1038/s41413-018-0016-9.
2. Zhao Y, Chen G-Y, Fang M. Research trends of rheumatoid arthritis and depression from 2019 to 2023: A bibliometric analysis. J Multidiscip Healthc. 2024;17:4465–74. https://doi.org/10.2147/JMDH.S478748.
3. Lima-Martínez MM, Campo E, Salazar J, Paoli M, Maldonado I, Acosta C, et al. Epicardial fat thickness as cardiovascular risk factor and therapeutic target in patients with rheumatoid arthritis treated with biological and non-biological therapies. Arthritis. 2014;2014:782850. https://doi.org/10.1155/2014/782850.
4. Johri N, Varshney S, Gandha S, Maurya A, Mittal P, Jangra S, et al. Association of cardiovascular risks in rheumatoid arthritis patients: Management, treatment and future perspectives. Health Sci Rev (Oxf). 2023;8:100108. https://doi.org/10.1016/j.hsr.2023.100108.
5. DeMizio DJ, Geraldino-Pardilla LB. Autoimmunity and inflammation link to cardiovascular disease risk in rheumatoid arthritis. Rheumatol Ther. 2020;7:19–33. https://doi.org/10.1007/s40744-019-00189-0.
6. Keleşoğlu Dinçer AB, Şahan HF. Increased epicardial adipose tissue thickness as a sign of subclinical atherosclerosis in patients with rheumatoid arthritis and its relationship with disease activity indices. Intern Emerg Med. 2024;19:1015–24. https://doi.org/10.1007/s11739-024-03542-6.
7. Kumar BS, Suneetha P, Mohan A, Kumar DP, Sarma KVS. Comparison of Disease Activity Score in 28 joints with ESR (DAS28), Clinical Disease Activity Index (CDAI), Health Assessment Questionnaire Disability Index (HAQ-DI) and Routine Assessment of Patient Index Data with 3 measures (RAPID3) for assessing disease activity in patients with rheumatoid arthritis at initial presentation. Indian J Med Res. 2017;146:S57–62. https://doi.org/10.4103/ijmr.IJMR_701_15.

8. Farhat H, Irfan H, Muthiah K, Pallipamu N, Taheri S, Thiagaraj SS, et al. Increased risk of cardiovascular diseases in rheumatoid arthritis: A systematic review. Cureus. 2022;14:e32308. https://doi.org/10.7759/cureus.32308.
9. Saha S, Singh R, Mir IA, Bansal N, Singh PK, Nadeem M. Epicardial fat thickness: A cardiometabolic risk marker in rheumatoid arthritis. Cureus. 2022;14:e21397. https://doi.org/10.7759/cureus.21397.
10. Wang T, Peng G, Liang H, Zhao B. Epicardial adipose tissue and left ventricular systolic function in rheumatoid arthritis assessed by two-dimensional speckle tracking echocardiography. Cureus. 2024;16:e60495. https://doi.org/10.7759/cureus.60495.
11. Argnani L, Zanetti A, Carrara G, Silvagni E, Guerrini G, Zambon A, et al. Rheumatoid arthritis and cardiovascular risk: Retrospective matched-cohort analysis based on the RECORD study of the Italian Society for Rheumatology. Front Med (Lausanne). 2021;8:745601. https://doi.org/10.3389/fmed.2021.745601.
12. Gastaldelli A, Kozakova M, Højlund K, Flyvbjerg A, Favuzzi A, Mitrakou A, et al. Fatty liver is associated with insulin resistance, risk of coronary heart disease, and early atherosclerosis in a large European population. Hepatology. 2009;49:1537–44. https://doi.org/10.1002/hep.22430.
13. Bal A, Ataman Ş, Bodur H, Rezvani A, Paker N, Tastekin N, et al. Characteristics of patients with rheumatoid arthritis in Turkey: Results from the Turkish League Against Rheumatism Rheumatoid Arthritis Registry. Arch Rheumatol. 2015;30:16–22. https://doi.org/10.5606/archrheumatol.2015.4224.
14. Resorlu H, Akbal A, Resorlu M, Gokmen F, Ates C, Uysal F, et al. Epicardial adipose tissue thickness in patients with ankylosing spondylitis. Clin Rheumatol. 2015;34:295–9. https://doi.org/10.1007/s10067-014-2568-4.
15. Lipson A, Alexopoulos N, Hartlage GR, Arepalli C, Oeser A, Bian A, et al. Epicardial adipose tissue is increased in patients with systemic lupus erythematosus. Atherosclerosis. 2012;223:389–93. https://doi.org/10.1016/j.atherosclerosis.2012.06.006.
16. Fatma E, Bunyamin K, Savas S, Mehmet U, Selma Y, Ismail B, et al. Epicardial fat thickness in patients with rheumatoid arthritis. Afr Health Sci. 2015;15:489–95. https://doi.org/10.4314/ahs.v15i2.23.
17. Alpaydın S, Buyukterzi Z, Akkurt HE, Yılmaz H. Impaired left ventricular diastolic functions and thickened epicardial adipose tissue in rheumatoid arthritis patients is correlated with DAS-28 score. Acta Cardiol Sin. 2017;33:182–7. https://doi.org/10.6515/acs20160608b.
18. Ilgun E, Kalkan AO, Ozdil H, Bilgi M, Demirayak M, Demir F, et al. Increased epicardial fat thickness in rheumatoid arthritis patients. Biomed Res. 2017;28:87–91.
19. Tarsitano MG, Pandozzi C, Muscogiuri G, Sironi S, Pujia A, Lenzi A, et al. Epicardial adipose tissue: A novel potential imaging marker of comorbidities caused by chronic inflammation. Nutrients. 2022;14:2926. https://doi.org/10.3390/nu14142926.
20. Packer M. Epicardial adipose tissue may mediate deleterious effects of obesity and inflammation on the myocardium. J Am Coll Cardiol. 2018;71:2360–72. https://doi.org/10.1016/j.jacc.2018.03.509.
21. Nagy E, Jermendy AL, Merkely B, Maurovich-Horvat P. Clinical importance of epicardial adipose tissue. Arch Med Sci. 2017;13:864–74. https://doi.org/10.5114/aoms.2016.63259.